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(for editorial comment, see page 176)

# Passive cigarette smoking and patients with asthma

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ABSTRACT: A study of the effect of passive smoking on patients with asthma is presented. Six patients were exposed for one hour to the air in a room in which tobacco smoke was produced mechanically over that period. The effects on symptoms, lung function and airways sensitivity to inhaled histamine were then measured and compared with the same patient's responses during a control day when they inhaled smoke-free air. **All six patients developed chest tightness and symptoms similar to an attack of asthma. The findings of respiratory and sensitivity tests suggest: (i) that passive smoking may trigger asthma attacks in subjects who suffer from asthma and (ii) that the airways of such subjects show increased histamine reactivity four hours after the passive smoke exposure.**

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PATIENTS WITH asthma who do not smoke commonly complain of "chest tightness" or wheezing as well as upper-respiratory tract symptoms when they are exposed to other people's cigarette smoke. This subjective response has been documented,[1-3] but objective studies of changes in lung function give conflicting results.[3,4]

A lack of effect of active cigarette smoking on the bronchial sensitivity to histamine and methacholine immediately after smoking a cigarette has been demonstrated.[5] Young asymptomatic smokers have been shown to be no more responsive to inhaled histamine than are non-smokers.[6] However, the effect of passive smoking on bronchial reactivity has not been studied in patients with asthma.

The aim of this study was to investigate the acute respiratory response in patients with mild to moderate asthma to passive cigarette smoking and to assess whether passive smoke inhalation alters bronchial sensitivity to inhaled histamine.

## Methods

Six subjects with mild to moderate asthma who were non-smokers were studied on an outpatient basis over two days. They were exposed to the air of a provocation room in which smoke was produced mechanically from one cigarette after another, continuously, over one hour. (Details of procedures and equipment used may be obtained from the writers on request.) The two-day study was organized as follows:

Day 1
- Baseline lung function
- Histamine inhalation test (HIT)
- Sixty minutes in room, cigarette not lit
- Repeat HIT four hours after leaving the room

Day 2
- Baseline lung function
- HIT
- Sixty minutes in room; cigarette lit
- Repeat HIT four hours after leaving room

The parameters of lung function chosen were the forced expiratory volume in one second ($FEV_1$), the vital capacity (VC) and the maximum mid-expiratory flow rate (MMEFR) with the same Vitalograph spirometer used for all measurements, and the peak expiratory flow rate (PEFR) measured by means of a Wright Peak Flow Meter. Measurements were taken at 15-minute intervals while in the provocation room, and afterwards until the readings had returned to baseline.

Each patient underwent four HITs during the study, and for each test the provocative concentration of histamine which produced a 20% fall in $FEV_1$ from a baseline value ($PC_{20}$) was calculated.

## Results

**Patient profiles are shown in Table 1. All six patients experienced some symptoms as a result of the passive smoking, eye irritation being the most common complaint. The four patients who gave a positive history of asthma attacks induced by passive smoking experienced "chest tightness" — of a mild degree for subjects 1 and 2 and of a moderate degree for subject 4 — and/or wheezing (subjects 4 and 5). Rhonchi (which had not been heard before the challenge) were heard on auscultation at the end of the exposure to smoke-contaminated air in subjects 4 and 5.**

The changes in $FEV_1$ (expressed as percentage change from the baseline) on both days are presented in Table 2. All

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six subjects showed falls in FEV, on the challenge day, the mean maximum fall being 11% compared with a mean maximum rise on the control day of 4.6%. These changes were statistically significant, according to a two-tailed paired *t*-test, in subjects 4, 5 and 6; subjects 1, 2 and 3 showed similar, though not statistically significant, changes. Similar trends were seen in VC, MMEFR and PEFR.

TABLE 1: Patient profiles

| | Subject number | | | | | |
|---|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 | 6 |
| Age (years) | 23 | 23 | 24 | 22 | 22 | 39 |
| Sex | M | M | M | F | M | F |
| Number of positive skin prick tests to 23 common allergens | 5 | 2 | 2 | 3 | 10 | 3 |
| IgE level (u/mL) | 140 | 43 | 1220 | 330 | 1140 | 310 |
| History of "chest tightness" or wheezing in response to passive smoking | Yes | Yes | No | Yes | Yes | No |

TABLE 2: Maximum variations of FEV, from baseline

| Subject Number | Control day | Challenge day | Significance |
|---|---|---|---|
| 1 | +1.3% | −8.5% | $P>0.05$ |
| 2 | +3.3% | −5.0% | $P>0.05$ |
| 3 | +5.1% | −1.7% | $P>0.05$ |
| 4 | +5.8% | −15.1% | $P<0.001$ |
| 5 | +2.8% | −26.9% | $P<0.055$ |
| 6 | +9.5% | −8.7% | $P<0.001$ |

TABLE 3: $PC_{20}$ values before entering and four hours after leaving the provocation room*

| | Subject number | | | | | |
|---|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 | 6 |
| Control day | | | | | | |
| $PC_{20}$ before | 4.39 | 6.96 | 1.60 | 0.86 | 0.39 | 0.74 |
| $PC_{20}$ after | 4.44 | 6.96 | 2.85 | 0.77 | 0.47 | 0.66 |
| Challenge day | | | | | | |
| $PC_{20}$ before | 4.59 | 7.21 | 0.92 | 0.90 | 0.22 | 1.00 |
| $PC_{20}$ after | 1.07 | 6.96 | 0.24 | 0.64 | 0.09 | 0.53 |
| $PC_{20}$ before/$PC_{20}$ after | | | | | | |
| Control day | 0.99 | 1.00 | 0.57 | 1.12 | 0.83 | 1.12 |
| Challenge day | 4.29 | 1.04 | 3.83 | 1.41 | 2.44 | 1.89 |

*$PC_{20}$ = provocative concentration of histamine (g/L) that produces a 20% fall in FEV, from a baseline value.

The results of the HITs are shown in Table 3. There was a trend for the $PC_{20}$ to fall after exposure to smoke to an extent which was not found on the control day. All but subject 2 had their lowest $PC_{20}$ at four hours after smoke provocation. The changes in $PC_{20}$ produced by passive smoking were statistically significant for the group, and indicate an increased airway irritability induced by passive smoking which was still detectable at four hours after cessation of exposure to ambient smoke.

## Discussion

Variable deteriorations in lung function parameters as a result of passive exposure to ambient cigarette smoke were found in all the subjects in our study. These deteriorations did not correlate with chest symptoms. In comparison with our findings, Dahms et al. observed a mean change of 21.4% in FEV, in 10 asthmatic subjects exposed passively to cigarette smoke.[4]

As a control day was included in the protocol, it is unlikely that the significant trend for the $PC_{20}$ to decrease which we observed at four hours after smoke exposure was due to diurnal variation or to variability in the test.

A four-hour interval between leaving the provocation room and performing the post-challenge HIT was chosen because animal studies have shown that neutrophil infiltration in response to airborne antigens is accompanied by an increase in histamine reactivity at four hours after a challenge.[7]

Thus our findings suggest that passive smoke inhalation may produce asthma attacks in subjects who suffer from asthma and may lead to increased bronchial reactivity to histamine for a time after such inhalation. Thus, the airways may be primed to react more vigorously to other triggers (for example, emotion, cold air, exercise) thereby initiating an attack which would not otherwise have occurred. This has obvious social implications for subjects with asthma who are frequently exposed to other people's cigarette smoke.

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